Treatment of Hypertropia with Relaxing Conjugate Vertical Prisms

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Certain selected cases of hypertropia can be permanently corrected with identical prisms placed base up over each eye. A child was first successfully treated by this method more than 14 years ago, and the excellent long-term outcome in her case, as well as generally encouraging results in a series of over 100 subsequent patients, suggest that in suitable cases the double vertical prism may be a useful addition to the methods available for the treatment of hypertropia.

Traditionally, prism glasses are used in the management of strabismus to neutralize the sensory consequences of the turn, and they are therapeutic in somewhat the same sense as refractive corrections, which improve function without eliminating the underlying deficiency. While clinical results may be excellent, they are achieved within the framework of preexisting motor and sensory patterns; thus in this respect, the conventional prism may be considered as essentially a compensatory or "relieving" device. In contrast, conjugate vertical prisms, used as described in this paper, have no effect upon the sensory angle between the two eyes, but instead are intended to alter the long-term course of motor adaptations in certain types of hypertropia. Because these prisms decrease the use of the paretic muscle and thus reduce progressive contralateral spasm and inhibitional palsy, they may be logically called "relaxing" prisms as distinguished from the "relieving" (or neutralizing) prisms as defined by Duke-Elder (1).

It is generally accepted that in cases of elevator insufficiency, constant fixation with the paretic eye may eventually result in habitual spasm and even permanent contractures of the yoke muscle. Similarly, inhibitional palsy of the contralateral antagonist may exaggerate the effect of slight weakness in the fixing eye so much that the original paresis may be obscured by secondary changes on the opposite side. When these mechanisms confuse the diagnosis, they may be temporarily excluded by patching the fixing eye. If conditions are still reversible, occlusion may decrease or even temporarily eliminate the squint. In contrast to its diagnostic value, such occlusion is of little benefit as a therapeutic measure because as soon as binocular use is resumed the patient will revert to his preferred fixation, and the circumstances leading to the secondary motor adaptation will become reestablished. However, if prisms can sufficiently minimize the use of the fixing eye in the field of maximum action of the paretic muscle, the recurrence of the conditions that determined the progression of the deviation may be avoided.

To the best of my knowledge, a survey of the literature has shown no reports of the systematic clinical application of this idea. Conjugate vertical prisms (base up or base down over both eyes) have been used infrequently in A&V syndromes, bilateral inferior rectus contractures, or certain types of nystagmus (2), but in the conventional way as compensatory devices to place the eyes into an optimum position for fusion or postural comfort with improvement in function only so long as the prisms con-

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tinue to be worn. In contradistinction, a similar correction prescribed as "relaxing" prisms has no immediate effect on measurements or symptoms, but over a prolonged course of time it exerts a cumulative influence on the underlying motor anomaly.

Prototype Case. The therapeutic rationale of relaxing conjugate vertical prisms is best explained in terms of the prototype case: the first and most successful clinical application of this method. The detailed history of this child can be considered as representative of the experience accumulated in the subsequent series of patients.

In August, 1957, a five-year-old girl was brought to the office because her left eye turned up and out to a conspicuous degree. Her mother also noticed that she closed the left eye in the bright sun and that the left eye became slightly "smaller" than the right when the child was especially tired. The initial examination showed an exotropia of 6 to 12 diopters and left hypertropia of 6 to 12 diopters. Both the horizontal and the vertical deviations varied markedly from day to day but were consistently greater for distance than for near. A slight ptosis on the left was also noted. The refractive error was insignificant, and visual acuity was 20/20 in each eye. The right hand and the right eye were strongly dominant. Ductions and versions at first seemed to be grossly normal in all directions of gaze. From the start it seemed that surgery would eventually be required, but the final decision was deferred until a series of repeated measurements could be collected over a period of time. Meanwhile, the child was referred to an orthoptist for further diagnostic studies and for antisuppression and fusion training. Conventional prism glasses with 2 diopters base up on the right and 2 diopters base down on the left were prescribed as a temporary measure.

After seven months, instrument fusion had improved considerably, but the amount of the deviation was essentially unchanged. A specific diagnosis of the muscles involved was still difficult. The left hypertropia was invariably greater with the right eye fixing than with the left eye fixing, and occasionally when the left eye fixed on a near object, a trace of right hyperphoria was observed. This would suggest that the deviation was primarily the result of a deficiency of an elevator on the right. However, the hypertropia was greatly increased on downward gaze, indicating paresis of a left depressor, and this possibility was supported by a positive Bielschowsky head tilt test. The Bielschowsky sign was absent when the child fixed on a near object with her left eye, and there was no habitual head tilt of the kind expected with true paresis of the left superior oblique in the presence of good vision and fusion. Furthermore, the left hypertropia was the same with eyes down and left as with eyes down and right, another finding that could not be explained on the basis of left superior oblique palsy alone. It was also puzzling that her conventional prism glasses were of no benefit but actually seemed to hinder fusion. With the glasses on, the child required an additional 3 or 4 diopters base up in the right cell of the stereoscope in order to fuse (the equivalent of 7 or 8 diopters of left hypertropia), but without the prism glasses she could fuse with only 1 to 3 diopters base up on the right. The visual acuity on the left had become very slightly subnormal, and even though objective and subjective angles seemed equal on the troposcope, the after image test indicated possible anomalous retinal correspondence both in the horizontal and vertical directions.

It appeared that secondary muscle spasm and inhibitional palsy might be important in this case. To clarify this situation, and also to correct the incipient amblyopia and the apparent tendency to abnormal correspondence, constant occlusion of the right eye was prescribed, and all binocular exercises were discontinued.

Several weeks later when the patch was removed, screen and parallax measurements showed: X4, LHO, X'O, and RH' trace. An increase of the hypertropia on downward gaze was no longer present. On the contrary, the turn was now greatest with the eyes elevated, and as expected with bilateral superior rectus palsy, there was left hypertropia on looking up and right and right hypertropia looking up and left. On the synoptophore the first measurement, immediately after removal of the patch, was X5, RH2. Within minutes the deviation changed into X5, LH2, and during the transition from the right to the left hyperphoria, the child spontaneously remarked that the picture tilted to one side and then righted itself at a different level. Occlusion was resumed and continued for a total of about six weeks until acuity had become bilaterally equal and all suggestion of anomalous correspondence had disappeared. At the end of this time no vertical deviation was detectable in the primary position, although there was 2 plus overaction of the left inferior oblique and 1 plus overaction of the right inferior oblique in their fields of maximum action. Nystagmoid movements were

seen in the fields of maximum action of both superior recti.

The patch was discontinued for one week, and left hypertropia of 10 diopters with XT6 for distance and left hypertropia of 8 diopters for near appeared again.

These findings seemed best explained on the basis of equal, or nearly equal, paresis of both superior recti. With the eyes relaxed, such equal weakness would result in no significant deviation, but the effort to fix would lead to spasm of the contralateral inferior oblique and inhibitional palsy of the contralateral superior oblique. With habitual preference of the right eye, the initially negligible deviation had become reinforced by the cumulative effects of continuous inhibition of the contralateral antagonist. Thus, the primary weakness of the superior recti had become masked by contralateral secondary changes, and the deviation had become dominated by the characteristics of the induced palsy of the left superior oblique including the positive Bielschowsky head tilt test and the accentuation of the hypertropia on downward gaze. The fact that the head tilt response was not always present, as well as the decrease or reversal of the left hypertropia when the child was forced to fix with her left eye, suggested that the weakness of the left superior oblique was the result of secondary changes. The nystagmoid movements on upward and outward gaze and the associated finding of slight ptosis on the left were also consistent with the diagnosis of superior rectus palsy. When prolonged fixation with the left eye was enforced by constant occlusion of the right eye, the inhibitional palsy of the left superior oblique and the secondary spasm of the left inferior oblique were relieved. A right hypertropia, resulting from underaction of the left superior rectus, was evident as long as left fixation was maintained. As soon as the child reverted to fixation with her right eye, the left hypertropia recurred almost immediately but, temporarily, to a lesser degree than before occlusion. It was now determined only by right superior rectus weakness, without the additional effect of the inhibitional palsy of the left superior oblique.

With the diagnosis reasonably clear, and acuity and retinal correspondence restored to normal, the child was considered ready for surgery which was planned for a convenient date several months later. Because of the strong preference of the right eye, it was decided to follow the usual procedure of correcting the secondary deviation with retroplacement of the left inferior obliques, more left than right.

While the child was being observed during the preoperative interval, the idea occurred that if bilateral base up prisms could decrease the use of the paretic superior rectus to a significant degree, the recurrence of the left hypertropia after removing occlusion might be prevented or at least delayed. Accordingly, the child's right eye was occluded again for three weeks until the vertical deviation in the primary position had again become negligible. Then, prisms of 4 diopters base up over each eye were prescribed to be used immediately and constantly after removal of the patch.

About one month later, the child returned quite remarkably improved. Screen and parallax measurements showed orthophoria for distance and near with or without the glasses. The Maddox rod showed right hyperphoria ¾ diopter with the left eye fixing and left hyperphoria ¾ diopter with the right eye fixing. In the primary position there was no exophoria, but with the eyes turned up or down 20 degrees at 6 meters there was an exophoria which averaged 10 diopters. Foveal fusion was present, although fusional amplitude was still rather low. The mother reported that the child's eyes always appeared straight with the prism glasses, but when she took them off, a slight inward turn of the left eye was occasionally noted, although spontaneous upward deviation was never seen again. There was still 2 plus overaction of the left inferior oblique and 1 plus overaction of the right inferior oblique on oblique upward gaze.

This was to have been the final preoperative visit, but even though the eyes were not absolutely perfect, the improvement was so striking that immediate surgery was no longer justified. The operation was deferred to permit a longer trial of the double vertical prisms. The child was to wear the prescription constantly, but no eye exercises were prescribed. When she was reexamined six months later, screen and parallax measurements showed no deviation either vertical or horizontal in the primary position at distance or near with or without the glasses. With the eyes turned up at 6 meters there was a 2 diopter exophoria, and with the eyes turned down at 6 meters there was a 4 diopter exophoria. Acuity was normal and equal. Fusional amplitude was considerably improved with prism convergence being 35 diopters at both distance and near. Not more than a

trace of overaction of the obliques was noted in their fields of maximum action.

In the attempt to be completely objective and honestly skeptical of this excellent result, an experienced orthoptist with no previous knowledge of the case was asked to verify these measurements with special attention to the vertical muscles. She also could find no vertical deviation either in the primary position or in the fields of maximum action of the obliques.

The prisms were continued for another five months, and in June, 1959, 11 months after the prism glasses were first prescribed, the child was examined and the following measurements were found: screen and parallax—X3 HO X'3 LH' ¾; eyes down and right—no abnormalities; eyes up and right—left inferior oblique 1 plus; eyes up and left—right inferior oblique 2 plus; eyes down and left—no abnormalities. There was no detectable deviation with the cover and uncover test, and there was good fusion with stereopsis. Gradual decrease in the wearing time of the glasses was suggested.

The child has now been followed for over 11 years without recurrence of the original problem. From time to time a very small and well compensated exophoria has appeared and rarely some negligible hyperphoria in extreme upward and oblique direction of gaze. But vision, foveal fusion, and absence of symptoms have been consistently maintained. At this time it certainly seems fair to say the result of treatment with the conjugate vertical prisms was at least as good as the best we could hope to achieve by surgical means.

Clinical Application. Subsequent to the prototype case, about 100 children have been treated according to this method with encouraging results. It has not been possible to duplicate the complete success of the original patient because children so perfectly suitable for this therapeutic approach are rare. However, in properly selected cases, and combined with appropriate conventional measures, the double vertical prism has proved to be a valuable supplement to the traditional methods of treating hypertropia.

The total number of children so treated is relatively small because the double vertical prism is indicated only under certain limited and relatively infrequent conditions. It was found that this form of therapy can be expected to benefit only the one type of deviation defined by the following criteria:

1. The paretic eye must be the fixing eye. There must be reason to suspect a relatively slight paresis of a vertical muscle with all, or the clinically significant portion, of the deviation being due to cumulative secondary changes on the opposite side.
2. Occlusion of the paretic eye must either eliminate (temporarily) the hypertropia in the primary position or reduce it to so small an amount that it can easily be controlled by fusion. If the secondary, as well as the primary, deviation with the eyes straight ahead becomes negligible after several weeks of patching, success with the double vertical prism is highly probable, and 4 to 6 diopters base up (for elevator paresis) over each eye may be prescribed for constant use immediately after removing the patch. This most favorable situation is well illustrated by the prototype case.
3. If occlusion reduces the hypertropia but leaves a residual deviation of 2 or, at the most, 3 diopters in the primary position, success is possible, but it may be wise to prescribe a combination of compensatory and therapeutic prisms. For example, 5 diopters base up on the right and 3 diopters base up on the left (in effect the sum of 4 diopters base up over each eye with one diopter base up right and one diopter base down left) may be used for right superior rectus paresis with 2 diopters of residual hypertropia after patching.
4. Finally, if occlusion does not decrease the deviation, or more than 2 or 3 diopters of turn remain, the double prism will probably be of limited value. Sometimes it may have a palliative effect, particularly in reducing the deviation prior to surgery and in postoperative patients with an incomplete surgical correction. Here again, the functions of therapeutic and compensatory prisms may be combined by placing all, or at least 4 diopters, of the necessary compensatory prism over the paretic eye. If the postoperative deviation represents a secondary innervational response that is slow to resolve, the appropriate prism may reinforce the effect of surgery in promoting the reversal of those persistent motor adaptations which have not yet progressed to structural changes. Occasional instances of clinical improvement in this situation have been en-

countered from time to time in the past when vertical prisms were placed over the paretic eye without any deliberate application of the principle of relaxing prisms. Urist (3) includes in his paper on vertical deviations a case successfully treated in much this manner. A child with right superior oblique palsy was left with 14 to 17 diopters of right hypertropia after recession of the ipsilateral inferior oblique for a much larger initial deviation. With 8 diopters prism base down over the paretic eye, the deviation gradually decreased, and after one and one-half years, the eyes were straight in the primary position. Urist concludes that the use of the prism postoperatively may have helped in the gradual relaxation of the secondary vertically deviating muscles, as we would expect on the basis of experience with conjugate vertical prisms.

Postoperative relaxing prisms may also be effectively utilized in a slightly different way as illustrated by the following case:

Case #2. B. B. was first seen in December, 1957, at the age of nine with alternating accommodative esotropia and double hypertropia of 20 diopters in each eye. After preliminary treatment with glasses and orthoptics, retroplacement of both inferior obliques 9 mm was carried out by Dr. Conrad Berens in April, 1958.

The early postoperative result was fairly good with the right hypertropia eliminated and the left deviation reduced to about 8 diopters of hyperphoria. However, the vertical deviation increased with time until in April, 1963, there were about 14 diopters of right and left hypertropia. She was alternating spontaneously, and on casual observation there was a cosmetically unacceptable seesaw movement of the eyes. Conventional neutralizing prisms had been tried without success. Four diopter base up prisms over each eye were then prescribed, and examination three months later showed no hypertropia in the primary position. There was slight regression over a period of time, and on her last visit in August, 1965, there was again a left hypertropia of 8 diopters but only on upward gaze, the eyes being straight in the primary position. There were no symptoms, and her appearance was satisfactory. In retrospect, however, the results might have been better if a base up prism had been used over the right eye immediately after surgery.

In typical cases, preliminary patching is absolutely essential for the most effective use of relaxing prisms. In the strictest sense, these prisms are prophylactic rather than truly therapeutic because they prevent recurrence of cumulative secondary changes which have already been relieved by the occlusion of the fixing paretic eye. Such patching may be omitted only if the vertical deviation is limited to upward gaze and absent or only intermittently present in the primary position. This category of patients includes children with primarily horizontal deviations when a small and inconstant vertical component seems to interfere with stable fusion.

It is perhaps in this group that conjugate prisms seem to have their greatest practical value. A vertical deviation so slight as to require no therapy if present alone, may become significant when it complicates a horizontal problem, and yet be too small to justify surgery and too variable to treat with conventional compensatory prisms. Here equal base up prisms may have an excellent stabilizing effect when combined with appropriate measures for the control of the horizontal component of the deviation.

Case #3. K. K., a four-year-old girl first seen in October, 1958, was typical of such patients. Alternating esotropia had been noticed by her mother since early infancy and had been unsuccessfully treated elsewhere with glasses worn only irregularly and "drops." On the initial visits screen and parallax showed ET 25, ET'35, LH 4–5 without glasses. Visual acuity was 20/20 in each eye, although the right eye was dominant. The left superior oblique appeared to be paretic, with a positive Bielschowsky test, and there was overaction of both inferior obliques. With correction of her hyperopia and plus 2.50 segments, measurements were E1, ET'14 through the distance correction, and EP'2 through her add. Left hypertropia of up to 5 diopters was intermittently present in the primary position. After occlusion of the right eye for two weeks, intermittent hypertropia with spasm of the left inferior oblique was still apparent.. But weakness of the left superior oblique could no longer be readily demonstrated, and the Bielschowsky test was now negative. These observations suggested that the apparent left superior oblique weakness might actually be inhibitional palsy derived from elevator paresis in the fixing right eye. The patch was removed, and three months later the Bielschowsky test was positive once more. Orthoptic therapy was prescribed, and glasses were continued. In October, 1959, her condition re-

mained essentially unchanged. The esotropia was partly controlled by the glasses, and fusion was somewhat improved but grossly apparent; vertical and horizontal deviation still occurred quite frequently, especially on upward gaze. At this time, 4 diopter prisms base up over each eye were incorporated in her glasses. On subsequent visits to the orthoptist, hypertropia in the primary position was never seen again although overaction of the left inferior oblique could still be demonstrated on looking up and right. Fusion and stereopsis continued to improve. She was last examined on August 29, 1967, after several months without any glasses, and measurements were as follows: acuity without correction 20/20 plus in each eye; screen and parallax O X′2; Maddox rod E2 O′. With maximum accommodation on fine print there was an esophoria of 10 diopters. No vertical deviation could be demonstrated even on oblique upward gaze. Glasses with plus 2.25 add were prescribed for prolonged study only.

One very small group of patients may possibly benefit from this method of treatment without occlusion and without regard to the foregoing principles of patient selection. We sometimes see a child with a large double hypertropia of 20 to 30 or more diopters in the primary position and spontaneous alternation so that under room conditions the eyes have a cosmetically disfiguring seesaw motion. These children are notoriously difficult to treat, and some may have repeated surgery to little avail. In four such cases, all of whom already had incompletely successful operations by various surgeons, equal prisms base up over each eye were empiricly tried and seemed to have a marked stabilizing effect. The amount of measurable turn did not necessarily change significantly, and often alternate covering continued to elicit as large a deviation as before, but under normal room conditions the eyes remained much more quiet and grossly straight. Regardless of measurements, these patients and their parents were delighted with the result and described it with such words as "the difference between night and day."

Unfortunately, in these cases the eventual complete removal of the prisms may not be possible. Two of these children, now young adults, have determined by trial and error that they can wear contact lenses (without prisms) up to about eight hours a day, but the base up prisms must apparently be used the rest of the time to prevent the recurrence of the seesaw phenomenon.

The theoretical considerations that indicate relaxing prisms in cases such as the prototype do not apply here, and it is admittedly most difficult to explain the action of the prisms in this special situation. However, from the practical standpoint, any evidence of improvement would seem to justify further clinical trial in these most difficult patients, especially since no adverse effects from conjugate vertical prisms have been observed. Even when such glasses prove ineffective, they are usually so well tolerated that there is no need to replace the lenses unless required for a change in refraction.

The useful role of double vertical prisms in the practical management of such complex deviation is shown by one of four patients mentioned above.

Case #4. C. R. was first seen in January, 1962, when she was 13 years old. She had a history of congenital nystagmus and myopia and esotropia first noted at the age of seven or eight months. Bilateral retroplacement of the medials had been done elsewhere in 1954, resulting in a very slight over correction which was being treated with orthoptics. On our initial visit she had 5 to 10 diopters of exotropia and a variable double hypertropia up to 30 diopters right and 40 diopters on the left. The right eye was dominant, and under ordinary room conditions the left hypertropia was prominent and cosmetically unacceptable. Four diopter prisms base up over each eye were incorporated in her refractive correction of $-8.50 = -1.50 \times 180$ right, and $-7.00 = -2.00 \times 175$ left, and orthoptic therapy was continued including fusion training as well as modified pleoptics for her slight amblyopia on the left. In May, 1962, the progress report from the orthoptist stated that the left hypertropia was seen less often, and the child's mother noticed marked improvement in her appearance at home. To a certain extent, this patient was her own control because subsequent attempts to remove the prisms resulted in recurrence of the left hypertropia, although the amount of the deviation was slowly decreasing over a prolonged period of time. In December, 1963, contact lenses were prescribed and worn eagerly, improving vision to about 20/25 right and 20/30 left. However, both the patient and her mother became aware that the left eye was again "floating" up with increasing frequency. She was, therefore, given glasses with a 5 diopter prism base up over each eye, but no refractive correction, to wear over her contacts as much as she found necessary to control the hypertropia. Use of these prisms for a few hours a day seemed to maintain the eyes in good position. When she was

last seen in October, 1968, the eyes were grossly straight, and there was peripheral fusion with stereopsis of 400 seconds of the arc. Screen and parallax showed 6 diopters exophoria and 3 diopters of left hyperphoria in the primary position. She reported that the prisms were necessary only occasionally, especially at times of fatigue or stress.

Discussion. The success of relaxing prisms in even a limited number of patients has interesting theoretical implications with respect to the pathogenesis of hypertropia, though the scope of this paper is admittedly confined to just one of the many well-known pathways the development of vertical deviation may take. In these special cases it appears that the habitual direction of gaze may play a leading role in the evolution of the squint. In such children the hypertropia may first become grossly evident or increase greatly at the age of two or three though it is thought to be the result of congenital anomaly. In early infancy, if the weakness of an elevator is very slight, and especially if it is balanced by a similar weakness on the other side, the eyes may be straight in the primary position and on downward gaze with deviation apparent only on looking up. Throughout most of a human life span, including early infancy, this condition would present little difficulty because of the relative unimportance of upward gaze. But when the child begins to crawl and walk, he suddenly discovers a world almost entirely above the level of his eyes, and he is encouraged to adopt an ocular posture which is unfavorable with respect to the field of action of the paretic muscle. These are the years of most intense challenge to upward gaze.

Although at first even the secondary deviation may be small enough to fall well within fusional range, if ocular dominance is established on the paretic side, chronic secondary motor adaptations may occur. With paresis of a superior rectus, the constant and prolonged overstimulation of the contralateral inferior oblique and under stimulation of the contralateral superior oblique may have cumulative and persistent effects that lead to ever increasing hypertropia of the non-fixing eye. Eventually the resulting chronic spasm of the yoke muscle and the persistent inhibitional palsy of its antagonist can dominate the clinical picture and be very difficult to distinguish from the originally negligible paresis of the opposite superior rectus, as in the prototype case. Initially the changes are reversible, and the hypertropia may decrease or entirely disappear for a short time after total occlusion of the fixing eye. Yet, as soon as binocular vision is again permitted, the secondary changes will occur as before.

As the child grows, his increasing height and periods spent in downward gaze on books, again ease the burden on the deficient elevators, and, occasionally, spontaneous improvement may occur. Yet, by this time the once reversible sensory and motor adaptations may have become firmly established. There may be permanent shortening and fibrosis of the contralateral inferior oblique and possibly foveal suppression with some degree of amblyopia. In this way an initially minor weakness of the superior rectus may in some cases develop into a substantial deviation that only surgery can correct, but if base up prisms can control the progress of these secondary changes and protect the normal development of stable fusion during the crucial years of upward gaze, the child might reach maturity with no clinically significant impairment of binocular vision.

Normal growth from kindergarten to the early teens raises a child's eyes by an amount roughly equivalent to the effect of 4 to 6 diopters at 20 feet. Thus if the prisms of this strength prove effective in controlling the hypertropia, we can reasonably hope that in due time the glasses can be removed with no recurrence of the vertical deviation. If a larger prism, that is, 10 to 15 diopters were required for the necessary effect, then normal growth would probably be insufficient to eliminate the need for this artificial method of lowering the eyes, and the patient would remain dependent on the prisms indefinitely. Such an outcome could hardly be considered as a complete cure. It was primarily for this reason that such larger prisms have not been tried, and the strength of the double base up correction was from the outset conservatively restricted to such a relatively small amount that at first it seemed improbable it could have any effect at all.

The same limitation applies to base down prisms which might logically be used for depressor paresis according to the same rationale as base up prisms are used for elevator paresis. It has been found that they may be effective to a certain extent, and improvement has been achieved this way in suitable cases, especially when the prisms are used as a supplement to other therapy. However, here normal growth intensifies the problem. The demands on the paretic depressor become greater rather than less with passage of time. Thus, the need for such prisms

is not outgrown, and no complete clinical cure can be expected by this method used alone.

Theoretically, it would be reasonable to weaken the base up prisms gradually as the child grows, but it has proved more practical to decrease the wearing time instead of reducing the strength of the glasses. The response to removing the correction is unpredictable, and if this step should prove premature, wearing time can be readjusted empiricly without the additional time and expense of changing the lenses.

Perhaps the most interesting aspect of the successful outcome of this method of treatment is how very minimal a change in the habitual position of the eyes may be sufficient to alter the mechanisms leading to the progression of the secondary deviation. It may seem that if so small a change in the customary direction of gaze can forestall the development of the squint, a child could accomplish the same thing for himself simply by raising his chin. Doubtless this sometimes occurs, but postural adjustment is determined by conditions as they currently exist and not in anticipation of events yet to come. If elevator palsy is too slight to cause subjective difficulty on upward gaze, there may be no stimulus for postural adjustment until the contralateral secondary changes have become well established. The head tilt that may ultimately develop will then reflect the new configuration rather than the original paresis. Perhaps there may be a rather narrow range of elevator weakness that can lead to this particular type of motor adaptation: The paresis must be great enough to cause significant innervational imbalance but not so great as to provoke immediate compensatory reaction through postural means.

The comprehensive papers of Urist (3, 4) on vertical muscle paresis include a very lucid discussion of the various secondary changes that can aggravate the consequences of simple vertical muscle weakness, including this particular pattern of motor adaptations that is presumed to take place in cases amenable to treatment with double base up prisms. He classifies as "Type 1" those children with evident deviation only in the field of action of the specific paretic muscle but no hypertropia in the primary position and no secondary contractures. This is equivalent to the condition that exists in a child like the prototype case immediately after a period of occlusion of the paretic eye. Urist found it a matter of some concern that such patients might eventually progress to the development of secondary contractures not yet present on initial examination, with hypertropia in the primary position as the ultimate result. He says, however, that after following these cases over the years, he did not see this happen.

Urist's experience in this matter is not necessarily incompatible with the less encouraging observations of this paper. In the susceptible child, secondary adaptations seem to appear quite rapidly, the recurrence of persistant inferior oblique spasm being a matter of weeks or days after occlusion of the paretic eye is stopped. Thus if the process is to take place at all, it is unlikely in the ordinary course of events that the child will be examined before the secondary changes are already well established. The elevator paresis in its original uncomplicated form may exist for so short a period in infancy that its presence as a minimal deviation limited to one particular direction of gaze would be impossible to evaluate with certainty in a child still too young for reliable fixation. Only by reversing the sequence of events by occlusion, say arranging an "instant replay" under direct observation, can the development of this type of heterotropia be witnessed as it takes place. The "Type 1" cases infrequently seen by Urist may represent those few children who are resistant for one reason or another to the cumulative and persistent secondary effects of fixation with the paretic eye.

The circumstances which determine whether a given patient will remain as "Type 1" or progress to another stage of strabismus are, as Urist also says, unknown. The degree of muscle weakness and whether fixation with the paretic eye is invariable or partly alternate may have some influence, and environmental factors must play at least some role. Although the relative importance of these conditions remains speculative, it seems sensible to try to limit the use of the eyes in the field of action of the paretic muscle and reasonable to advise the parents of such patients to discourage upward gaze when this can be done without undue disruption of the normal routines of life. Television viewing from the floor can be forbidden, easels and blackboards can be adjusted to eye level, and seating at the back of the classroom can be requested.

Evidence that minimal changes in habitual direction of gaze may have marked effects in certain cases of vertical muscle imbalance also has bearing on the general principles of prism use. It is necessary to reconsider the usual convention of dividing compensatory prisms into base up on one side and base down on the other. There are obvious practical advantages to splitting the prism, and frequently this

remains the procedure of choice. But if the paretic eye is preferred for fixation, and spasm of the yoke muscle is prominent, it may be best to use the entire prism on the paretic eye. Experience with conjugate base up prisms has definitely indicated that 4 prism diopters base up on the right, for example, is by no means always identical and interchangeable in effect with 2 diopters base up on the right and 2 diopters base down on the left. Obviously a given prism can move the eye or move the retinal image or move both, depending on how it is placed in relation to the dominant eye. If the prism is put over the fixing eye, the eye will follow the apparent displacement of the object of regard, and the shift of the contralateral retinal image will be brought about by a conjugate movement on that side. In contrast, if the prism is put over the deviating eye, it moves the retinal image on that side without a change in the position of the eyes, unless a small fusion movement takes place. Both reactions occur with the split prism. Thus, 2 prism diopters base up on a dominant right eye and 2 diopters base down on the left would neutralize a 4 diopter hypertropia but also lower both eyes by two diopters. Similarly, if the entire 4 diopter correction were used base up on the right, both eyes would be depressed to a corresponding degree. In contrast, 4 diopters of prism base down on the left would compensate for the deviation by moving the retinal image on that side with no direct effect on the position of the eyes and could thus be considered as having a neutral motor influence.

Although these differences seem negligible in amount, they cannot be overlooked when it has been found that even minor changes in the habitual direction of gaze may have significant long-term consequences in susceptible patients. It is not uncommon for an angle of vertical deviation to enlarge with the use of a compensatory prism correction. It is possible that in some of these cases secondary contractures and inhibitional palsy are truly increased by an inadvertently unfavorable arrangement of prisms which imposes additional innervating stress in the fields of the paretic muscles.

The immediate increase in hypertropia that occurred in the prototype patient described in this paper when a 4 diopter compensatory prism was equally split between the two eyes might possibly be explained as the result of lowering the right eye by 2 diopters while the marked inhibitional palsy of the contralateral superior oblique restrained adequate conjugate movement on the left.

Whenever a relieving prism is placed over the fixing eye, sensory neutralization of the deviation is complicated by a small shift in binocular posture which may be either advantageous or detrimental depending on the specific configuration of the motor imbalance. Thus it appears that in prescribing prisms for any purpose, not only the net strength of the combination but its distribution between the two eyes may be of great clinical importance.

Summary. Bilateral base up prisms can permanently correct hypertropia in selected patients. It appears that in such cases the deviation develops in consequence of chronic contralateral muscle changes induced by habitual use of the fixing eye in the field of maximum action of a paretic elevator. If hypertropia is temporarily relieved by occlusion of the fixing eye, appropriate prisms may sufficiently decrease the demands on the paretic muscle to prevent recurrent deviation when binocular vision is resumed. When occlusion only partly reduces hypertropia, prisms to limit gaze in the field of the paretic muscle may be advantageously combined with a neutralizing correction for the residual turn. The results obtained with double vertical prisms suggest that habitual direction of gaze may play an important part in the development of certain vertical deviations.

Author's note: The most important portions of this work were done while the author was privileged to be associated with the late Dr. Conrad Berens, whose indispensable encouragement and interest in this project are acknowledged with deepest gratitude. The original version of this paper was read and criticized by Dr. Berens not long before his untimely death in 1963. The present revision, incorporating more recent data, was presented in part to the meeting of the Eastern Section of the American Association of Certified Orthoptists at Nassau Hospital, Mineola, New York, April 29, 1969.

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